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L9	L8 and l1	15	L9
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L5	L4 and methotrexeate	0	L5
L4	LDL adj (Apo B receptor)	1561	L4
L3	L2 and (U937 cells)	13915	L3
L2	L1 and RPMI	13923	L2
L1	foetal calf serum or fetal calf serum	100214	L1

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☐ 1. Document ID: US 6670452 B2

L9: Entry 1 of 15 File: USPT Dec 30, 2003

US-PAT-NO: 6670452
DOCUMENT-IDENTIFIER: US 6670452 B2

TITLE: Non-naturally occurring lipoprotein particle

DATE-ISSUED: December 30, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Halbert; Gavin William	Jordanhill			GB
Owens; Moira Doreen	Shawlands			GB
Baillie; George	Kilmarnock			GB

US-CL-CURRENT: 530/359; 435/7.1, 530/350, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWMC | Draw De

☐ 2. Document ID: US RE37891 E

L9: Entry 2 of 15 File: USPT Oct 22, 2002

US-PAT-NO: RE37891
DOCUMENT-IDENTIFIER: US RE37891 E

TITLE: Target and background capture methods with amplification for affinity assays

DATE-ISSUED: October 22, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Collins; Mark L.	Holden	MA		
Halbert; Donald N.	Milford	MA		
King; Walter	Maynard	MA		
Lawrie; Jonathan M.	Milford	MA		

US-CL-CURRENT: 435/6; 435/15, 435/174, 435/5, 435/7.1, 435/91.2, 536/24.3, 536/24.32, 536/24.33, 536/25.4

h e b b g e e e f e f e ef b e

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 3. Document ID: US 6331542 B1

L9: Entry 3 of 15 File: USPT Dec 18, 2001

US-PAT-NO: 6331542
DOCUMENT-IDENTIFIER: US 6331542 B1

TITLE: Protease inhibitors

DATE-ISSUED: December 18, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Carr; Thomas Joseph	Phoenixville	PA		
Desjarlais; Renee Louise	St. Davids	PA		
Gallagher; Timothy Francis	Harleysville	PA		
Halbert; Stacie Marie	Harleysville	PA		
Oh; Hye-Ja	Exton	PA		
Thompson; Scott Kevin	Phoenixville	PA		
Veber; Daniel Frank	Ambler	PA		
Yamashita; Dennis Shinji	King of Prussia	PA		
Yen; Jack Hwekwo	Malvern	PA		

US-CL-CURRENT: 514/237.8; 514/332, 514/357, 514/423, 514/482, 514/483, 514/590, 544/162, 546/265, 546/332, 548/531, 560/13, 560/159, 560/22, 560/25, 564/35

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 4. Document ID: US 6284777 B1

L9: Entry 4 of 15 File: USPT Sep 4, 2001

US-PAT-NO: 6284777
DOCUMENT-IDENTIFIER: US 6284777 B1

TITLE: Carbohydrazide protease inhibitors

DATE-ISSUED: September 4, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Halbert; Stacie Marie	Harleysville	PA		
Thompson; Scott Kevin	Phoenixville	PA		

US-CL-CURRENT: 514/332; 514/357, 514/482, 514/590, 546/265, 546/332, 560/159, 560/22, 564/35

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☐ 5. Document ID: US 6265176 B1

L9: Entry 5 of 15 File: USPT Jul 24, 2001

US-PAT-NO: 6265176
DOCUMENT-IDENTIFIER: US 6265176 B1

TITLE: Dot immunoassay on plastic sheets

DATE-ISSUED: July 24, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lin; Tsue-Ming	Miami	FL		
Halbert; Seymour P.	Miami	FL		

US-CL-CURRENT: 435/7.92; 422/56, 422/57, 427/2.11, 435/5, 435/7.94, 435/7.95, 435/970, 435/975, 436/518, 436/531, 436/809

☐ 6. Document ID: US 6232342 B1

L9: Entry 6 of 15 File: USPT May 15, 2001

US-PAT-NO: 6232342
DOCUMENT-IDENTIFIER: US 6232342 B1

TITLE: Protease inhibitors

DATE-ISSUED: May 15, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Carr; Thomas Joseph	Phoenixville	PA		
Desjarlais; Renee Louise	St. Davids	PA		
Gallagher; Timothy Francis	Harleysville	PA		
Halbert; Stacie Marie	Harleysville	PA		
Oh; Hye-Ja	Exton	PA		
Thompson; Scott Kevin	Phoenixville	PA		
Veber; Daniel Frank	Ambler	PA		
Yamashita; Dennis Shinji	King of Prussia	PA		
Yen; Jack Hwekwo	Malvern	PA		

US-CL-CURRENT: 514/524; 514/602, 558/390, 564/82

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☐ 7. Document ID: US 5998470 A

L9: Entry 7 of 15 File: USPT Dec 7, 1999

US-PAT-NO: 5998470
DOCUMENT-IDENTIFIER: US 5998470 A

TITLE: Protease inhibitors

DATE-ISSUED: December 7, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Halbert; Stacie Marie	Harleysville	PA		
Thompson; Scott Kevin	Phoenixville	PA		
Veber; Daniel Frank	Ambler	PA		

US-CL-CURRENT: 514/482; 530/300, 530/331, 560/158

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWMC | Draw De

☐ 8. Document ID: US 5955492 A

L9: Entry 8 of 15 File: USPT Sep 21, 1999

US-PAT-NO: 5955492
DOCUMENT-IDENTIFIER: US 5955492 A

TITLE: Carboxylic acid indole inhibitors of chemokines

DATE-ISSUED: September 21, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thompson; Scott K.	Phoenixville	PA		
Halbert; Stacie M.	Harleysville	PA		
Widdowson; Katherine L.	King of Prussia	PA		

US-CL-CURRENT: 514/419; 514/382, 514/784, 514/826, 514/863, 548/250, 548/252, 548/254, 548/490, 548/491, 548/494, 549/440, 562/405, 562/466, 562/468

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWMC | Draw De

☐ 9. Document ID: US 5750338 A

L9: Entry 9 of 15 File: USPT May 12, 1998

US-PAT-NO: 5750338
DOCUMENT-IDENTIFIER: US 5750338 A

h e b b g e e e f e f e ef b e

** See image for Certificate of Correction **

TITLE: Target and background capture methods with amplification for affinity assays

DATE-ISSUED: May 12, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Collins; Mark L.	Holden	MA		
Halbert; Donald N.	Milford	MA		
King; Walter	Maynard	MA		
Lawrie; Jonathan M.	Milford	MA		

US-CL-CURRENT: 435/6; 435/174, 435/5, 435/7.1, 435/91.2, 536/24.3, 536/24.32, 536/24.33

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWMC | Draw De

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☐ 10. Document ID: US 5684032 A

L9: Entry 10 of 15 File: USPT Nov 4, 1997

US-PAT-NO: 5684032
DOCUMENT-IDENTIFIER: US 5684032 A

TITLE: Compounds

DATE-ISSUED: November 4, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Elliott; John Duncan	Wayne	PA		
Leber; Jack Dale	Doylestown	PA		
Thompson; Scott Kevin	Phoenixville	PA		
Halbert; Stacie Marie	Harleysville	PA		

US-CL-CURRENT: 514/414; 514/419, 548/454, 548/491

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWMC | Draw De

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L3	L2 and (U937 cells)	13915	L3
L2	L1 and RPMI	13923	L2
L1	foetal calf serum or fetal calf serum	100214	L1

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DOCUMENT-IDENTIFIER: US 6831105 B2
TITLE: Compositions comprising ether compounds and pharmaceutical uses therefor

Brief Summary Text (12):
Primarily, the liver takes up and degrades circulating cholesterol to bile acids, which are the end products of cholesterol metabolism. The uptake of cholesterol-containing particles is mediated by LDL receptors, which are present in high concentrations on hepatocytes. The LDL receptor binds both apo E and apo B-100 and is responsible for binding and removing both IDL and LDL from the circulation. IN addition, remnant receptors are responsible for clearing chylomicrons and VLDL remnants i.e., IDL). However, the affinity of apo E for the LDL receptor is greater than that of apo B-100. As a result, the LDL particles have a much longer circulating life span than IDL particles; LDL circulates for an average of two and a half days before binding to the LDL receptors in the liver and other tissues. High serum levels of LDL, the "bad" cholesterol, are positively associated with coronary heart disease. For example, in atherosclerosis, cholesterol derived from circulating LDL accumulates in the walls of arteries. This accumulation forms bulky plaques that inhibit the flow of blood until a clot eventually forms, obstructing an artery and causing a heart attack or stroke.

Brief Summary Text (13):
Ultimately, the amount of intracellular cholesterol liberated from the LDL controls cellular cholesterol metabolism. The accumulation of cellular cholesterol derived from VLDL and LDL controls three processes. First, it reduces the cell's ability to make its own cholesterol by turning off the synthesis of HMGCoA reductase, a key enzyme in the cholesterol biosynthetic pathway. Second, the incoming LDL-derived cholesterol promotes storage of cholesterol by the action of ACAT, the cellular enzyme that converts cholesterol into cholesteryl esters that are deposited in storage droplets. Third, the accumulation of cholesterol within the cell drives a feedback mechanism that inhibits cellular synthesis of new LDL receptors. Cells, therefore, adjust their complement of LDL receptors so that enough cholesterol is brought in to meet their metabolic needs, without overloading (for a review, see Brown & Goldstein, In, The Pharmacological Basis Of Therapeutics, 8th Ed., Goodman & Gilman, Pergaman Press, NY, 1990, Ch. 36, pp. 874-896).

Brief Summary Text (14):
High levels of apo B-containing lipoproteins can be trapped in the subendothelial space of an artery and undergo oxidation. The oxidized lipoprotein is recognized by scavenger receptors on macrophages. Binding of oxidized lipoprotein to the scavenger receptors can enrich the macrophages with cholesterol and cholesteryl esters independently of the LDL receptor. Macrophages can also produce cholesteryl esters by the action of ACAT. LDL can also be complexed to a high molecular weight glycoprotein called apolipoprotein(a), also known as apo(a), through a disulfide bridge. The LDL-apo(a) complex is known as Lipoprotein(a) or Lp(a). Elevated levels of Lp(a) are detrimental, having been associated with atherosclerosis, coronary heart disease, myocardial infarcation, stroke, cerebral infarction, and restenosis following angioplasty.

Brief Summary Text (31):
The statins are inhibitors of cholesterol synthesis. Sometimes, the statins are used in combination therapy with bile-acid-binding resins. Lovastatin (MEVACOR, Merck & Co., Inc.), a natural product derived from a strain of Aspergillus; pravastatin (PRAVACHOL, Bristol-Myers Squibb Co.); and atorvastatin (LIPITOR, Warner Lambert) block cholesterol synthesis by inhibiting HMGCoA, the key enzyme involved in the cholesterol biosynthetic pathway. Lovastatin significantly reduces serum cholesterol and LDL-serum levels. It also slows progression of coronary atherosclerosis. However, serum HDL levels are only slightly increased following lovastatin administration. The mechanism of the

LDL-lowering effect may involve both reduction of VLDL concentration and induction of cellular expression of LDL-receptor, leading to reduced production and/or increased catabolism of LDL. Side effects, including liver and kidney dysfunction are associated with the use of these drugs.

Drawing Description Text (18):
FIG. 17 shows the effect on the serum lipoprotein cholesterol profile of LDL receptor deficient mice following seven daily treatments with Compound A.

Detailed Description Text (256):
The present compositions can be administered together with treatment with irradiation or one or more chemotherapeutic agents. For irridiation treatment, the irradiation can be gamma rays or X-rays. For a general overview of radiation therapy, see Hellman, Chapter 12: Principles of Radiation Therapy Cancer, in: Principles and Practice of Oncology, DeVita et al., eds., 2nd. Ed., J.B. Lippencott Company, Philadelphia. Useful chemotherapeutic agents include methotrexate, taxol, mercaptopurine, thioguanine, hydroxyurea, cytarabine, cyclophosphamide, ifosfamide, nitrosoureas, cisplatin, carboplatin, mitomycin, dacarbazine, procarbizine, etoposides, campathecins, bleomycin, doxorubicin, idarubicin, daunorubicin, dactinomycin, plicamycin, mitoxantrone, asparaginase, vinblastine, vincristine, vinorelbine, paclitaxel, and docetaxel. In a specific embodiment, a composition of the invention further comprises one or more chemotherapeutic agents and/or is administered concurrently with radiation therapy. In another specific embodiment, chemotherapy or radiation therapy is administered prior or subsequent to administration of a present composition, preferably at least an hour, five hours, 12 hours, a day, a week, a month, more preferably several months (e.g., up to three months), subsequent to administration of a composition of the invention.

Detailed Description Text (307):
10. Example: Effect of Compound A on Lipoprotein Cholesterol Profile in LDL Receptor-Deficient Mice

Detailed Description Text (308):
Homozygous familial hypercholesterolemia is a rare human disease (.about.1/1,000,000) characterized by absent or defective LDL receptors, markedly elevated serum LDL cholesterol levels and very early and severe onset of atherosclerosis. The more common form of this disease in humans, heterozygous familial hypercholesterolemia, occurs in about one in every 500 humans. Patients with the heterozygous form of this disease also present with elevated LDL levels and early onset of atherosclerosis.

Detailed Description Text (309):
The effect of Compound A on LDL levels in a murine model of homozygous familial hypercholesterolemia (Ishibashi et al., 1993, J. Clin. Invest. 92:883-893; Ishibashi et al., 1994, J. Clin. Invest. 93:1885-1893) was studied. LDL receptor-deficient mice have elevated LDL cholesterol relative to wild type mice when fed a chow diet. When fed cholesterol-enriched diets, these mice develop atherosclerosis.

Detailed Description Text (310):
FIG. 17 shows the lipoprotein cholesterol profiles (Bisgaier et al., J. Lipid Res. 38:2502-2515) of 4 chow-fed female LDL receptor deficient mice prior to and following therapy with 300 mg/kg/day of Compound A. All mice showed a rapid and significant reduction in LDL cholesterol after one week of treatment. In addition, FIG. 17 shows that Compound A caused HDL elevation in all treated mice.

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